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L1          103 S IBD? AND LACTOFERRIN?
L2            5 S (IBD TREATMENT) AND MONITOR?
L3            0 S L2 AND L1
L4            3 DUPLICATE REMOVE L2 (2 DUPLICATES REMOVED)
L5            0 S L4 AND LACTOF?
L6           20 S L1 AND TREATMENT?
L7           13 DUPLICATE REMOVE L6 (7 DUPLICATES REMOVED)
L8            3 S L7 AND PD<2001
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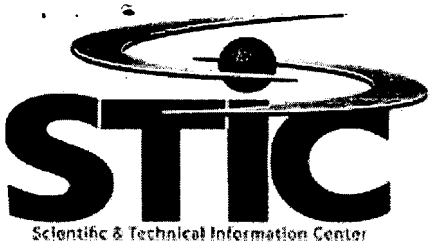

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AN 2000365992 EMBASE

TI Enteric bacteria, lipopolysaccharides and related cytokines in inflammatory bowel disease: Biological and clinical significance.

AU Caradonna L.; Amati L.; Magrone T.; Pellegrino N.M.; Jirillo E.; Caccavo D.

CS Dr. E. Jirillo, Immunologia, Policlinico, Piazza G. Cesare 4, 70124 Bari, Italy. jirillo@midim.uniba.it

SO Journal of Endotoxin Research, (2000) Vol. 6, No. 3, pp. 205-214. .
Refs: 126
ISSN: 0968-0519 CODEN: JENREB

CY United Kingdom

DT Journal; General Review

FS 004 Microbiology
026 Immunology, Serology and Transplantation
030 Pharmacology
037 Drug Literature Index
048 Gastroenterology

LA English

SL English

ED Entered STN: 2 Nov 2000
Last Updated on STN: 2 Nov 2000

AB Ulcerative colitis (UC) and Crohn's disease (CD) [inflammatory bowel disease (IBD)] are both characterized by an exaggerated immune response at the gut associated lymphoreticular tissue level. Such an abnormal and dysregulated immune response may be directed against luminal and/or enterio bacterial antigens, as also supported by murine models of inflammatory bowel disease (IBD) caused by organisms such as Citrobacter rodentium and Helicobacter hepaticus. Bacterial endotoxins or lipopolysaccharides (LPS) have been detected in the plasma of IBD patients and an abnormal microflora and/or an increased permeability of the intestinal mucosa have been invoked as cofactors responsible for endotoxemia. At the same time, the evidence that phagocytosis and killing exerted by polymorphonuclear cells and monocytes and the T-cell dependent antibacterial' activity are decreased in IBD patients may also explain the origin of LPS in these diseases. In IBD, pro-inflammatory cytokines and chemokines have been detected in elevated amounts in mucosal tissue and/or in peripheral blood, thus suggesting a monocyte/macrophage stimulation by enteric bacteria and/or their constituents (e.g. LPS). On these grounds, in experimental models and in human IBD, anti-cytokine monoclonal antibodies and interleukin receptor antagonists are under investigation for their capacity to neutralize the noxious effects of immune mediators. Finally, the administration of lactobacilli is beneficial in human IBD and, in murine colitis, this treatment leads to a normalization of intestinal flora, reducing the number of colonic mucosal adherent and translocated bacteria.

CT Medical Descriptors:
*Enterobacteriaceae
*enteritis
ulcerative colitis
Crohn disease
immune response
reticuloendothelial system
immunoregulation
Citrobacter
Helicobacter hepaticus
toxin analysis
intestine mucosa permeability
intestine flora
endotoxemia
phagocytosis

polymorphonuclear cell
monocyte
T lymphocyte
antibacterial activity
macrophage
cell stimulation
Lactobacillus
bacterial translocation
bacterium adherence
human
nonhuman
mouse
animal experiment
animal model
controlled study
human cell
animal cell
review
Drug Descriptors:
*bacterium lipopolysaccharide: EC, endogenous compound
*cytokine: EC, endogenous compound
bacterial antigen: EC, endogenous compound
endotoxin: EC, endogenous compound
chemokine: EC, endogenous compound
interleukin receptor: EC, endogenous compound
interleukin 10: EC, endogenous compound
interleukin 12: EC, endogenous compound
gamma interferon: EC, endogenous compound
CD4 antigen: EC, endogenous compound
CD8 antigen: EC, endogenous compound
tumor necrosis factor alpha: EC, endogenous compound
interleukin 8: EC, endogenous compound
monocyte chemotactic protein 1: EC, endogenous compound
granulocyte macrophage colony stimulating factor: EC, endogenous compound
butyric acid: EC, endogenous compound
interleukin 1beta: EC, endogenous compound
immunoglobulin A: EC, endogenous compound
lactoferrin: EC, endogenous compound
glyceraldehyde 3 phosphate: EC, endogenous compound
nitric oxide: EC, endogenous compound
monoclonal antibody: PD, pharmacology
monoclonal antibody ca2: PD, pharmacology
tumor necrosis factor alpha antibody: PD, pharmacology
cytokine antibody: PD, pharmacology
CD45 antigen: EC, endogenous compound
recombinant interleukin 10: PD, pharmacology
placebo
antisense oligonucleotide: PD, pharmacology
immunoglobulin enhancer binding protein: EC, endogenous compound
unclassified drug
RN (interleukin 12) 138415-13-1; (gamma interferon) 82115-62-6; (interleukin 8) 114308-91-7; (butyric acid) 107-92-6, 156-54-7, 461-55-2; (lactoferrin) 55599-62-7; (glyceraldehyde 3 phosphate) 142-10-9; (nitric oxide) 10102-43-9
CN Cdp 571